Dissertation on

COMPARISON OF INDUCTION AND RECOVERY CHARACTERISTICS OF PROPOFOL AND SEVOFLURANE IN DAY CARE ADULT TONSILLECTOMIES

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CERTIFICATE

This is to certify that the dissertation entitled, **"COMPARISON OF INDUCTION AND RECOVERY CHARACTERISTICS OF PROPOFOL AND SEVOFLURANE IN DAY CARE ADULT TONSILLECTOMIES"** submitted by Dr.Ahila.R , in partial fulfillment for the award of the degree of Doctor of Medicine in Anaesthesiology by the Tamilnadu Dr.M.G.R. Medical University, Chennai is a bonafide record of the work done by her in the Department of Anaesthesiology , Madras Medical College, during the academic year 2005 – 2008.

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INTRODUCTION

Ambulatory anaesthesia is one administered for elective surgical procedure performed on carefully selected patients, which is undertaken with all its constituent elements (admission, surgery and discharge home) on the same day. It is also referred to as day case, day care or outpatient anaesthesia and more recently office - based anaesthesia.

Ambulatory anaesthesia is a rapidly growing subspecialty. Although its history is as old as the history of general anaesthesia itself, it has emerged as a recognized concept and is evolving over the past couple of decades.

In the US, it comprises 70 percent of anaesthesia services provided. In the UK, the NHS plan, published recently predicts that 75 percent of elective surgical procedures will soon be conducted as day cases. Back home, 70 percent of elective surgeries that qualify the criteria are performed as day cases in Hinduja Hospital, Mumbai.

Anaesthetic agents today have been designed and marketed to meet specific niche criteria for ambulatory anaesthesia. Among the agents available in India, propofol and sevoflurane have increased the ability of the anaesthesiologist to provide a successful day case experience.

The present study compares the induction and recovery characteristics of these two anaesthetic drugs and their usefulness in ambulatory anaesthesia.

AIM OF THE STUDY

The aim of the study is to compare the induction and recovery characteristics of propofol and sevoflurane by the time to loss of consciousness, incidence of apnoea, induction complications, recovery times and incidence of postoperative nausea, vomiting & pain when they are used as sole induction and maintenance anaesthetic agent in adult tonsillectomies.

AMBULATORY ANAESTHESIA

In the early 1900s, an American anaesthesiologist, Ralph Waters conceptualized ambulatory anaesthesia. There was little interest in ambulatory surgical care until the late 1960s, when the first hospital based ambulatory surgical units were developed. Over the last two decades, outpatient surgery has grown at an exponential rate, progressing from the practice of performing simple procedures on healthy outpatients to encompassing a broad spectrum of patient care in freestanding ambulatory surgery centers.

The surgery may be done in a hospital, a freestanding surgery centre or in some cases, a surgeon's office. Anaesthesia care is given or supervised by anaesthesiologist.

The advantages of lower cost, lower rate of hospital acquired infection, less separation of patients from their home and family environment, less patient anxiety and greater patient convenience have been demonstrated by this sub-specialty over a period of five decades. It was well established that paediatric patients recovered better at home without separating from their mothers.

Patients save money by less preoperative laboratory tests and fewer post operative medications and by recovering at home. They are continued to be employed while recuperating, thus beds are free for the hospital for sicker patients and for emergency surgeries. Patients have the greater flexibility in selecting the time of their operation.

Newer anaesthetic drugs allow patients to recover faster, permitting the number and the complexity of cases to include longer and more complex procedures, permitting a safer operation theatre without flammable anaesthetics. (Appropriate pain management and prophylaxis for PONV is included as part of the discharge planning.)

Technology has offered sophisticated monitors to monitor patients more carefully during anaesthesia, thus permitting sicker patients with more challenging medical conditions to be considered for ambulatory anaesthesia.

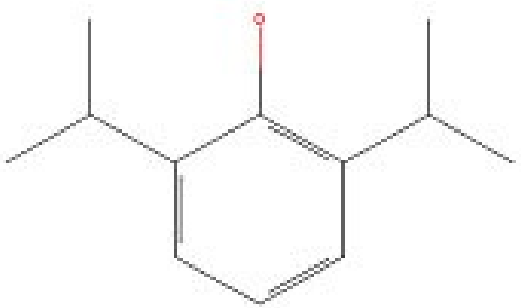
There are several types of anaesthetic techniques available for ambulatory surgery ranging from local anaesthesia to general anaesthesia. The anaesthetic technique recommended depends on several factors like the surgical procedure, the medical history of the patient and the patient's preference.

The four anaesthetic options are:

- General anaesthesia
- Regional anaesthesia
- Monitored anaesthesia care
- Local anaesthesia

General anaesthesia with regional anaesthesia for post operative pain relief is an ideal combination as it combines the advantages of both the comfort and lack of awareness in the former and the good quality of pain relief with the later.

PHARMACOLOGY OF PROPOFOL



Propofol is a substituted isopropyl phenol (2, 6 - di isopropyl phenol) that is administered intravenously as 1 % solution in an aqueous solution of 10 % soybean oil, 2.25% glycerol and 1.2% purified egg phosphatide. This drug is chemically distinct from all other drugs that act as intravenous sedative - hypnotics.

Administration of propofol, 1.5 to 2.5 mg/kg as a rapid IV injection (<15 secs), produces unconsciousness within about 30 seconds. Awakening is more rapid and complete than that after induction of anaesthesia with all other drugs.

The more rapid return of consciousness with minimal residual central nervous system effects is one of the most important advantages of propofol.

MECHANISM OF ACTION

Propofol is presumed to exert it's sedative - hypnotic effects through an interaction with GABA receptor, the principal inhibitory neurotransmitter in the central nervous system. When GABA receptor is activated, transmembrane chloride conductance increases, resulting in hyperpolarisation of the post synaptic cell membrane and functional inhibition of the postsynaptic neuron. The interaction of propofol with specific components of $GABA_A$ receptors appears to decrease the rate of dissociation of the inhibitory neurotransmitter, GABA from the receptor, thereby increasing the duration of the GABA activated opening of the chloride channel with resulting hyper polarization of cell membranes.

PHARMACOKINETICS

Hepatic metabolism and tissue uptake (possibly into the lungs) are both important in removal of this drug from the plasma. Hepatic metabolism is rapid and extensive, resulting in inactive, water - soluble sulphate and glucuronic acid conjugates that are excreted by the kidneys. The elimination half time is 0.5 to 1.5 hours, but more important, the context - sensitive half time for propofol infusions lasting up to 8 hours is < 40 minutes. The context - sensitive half time of propofol is minimally influenced by the duration of infusion because of rapid metabolic clearance when the infusion is discontinued, such that drug that returns from tissue storage sites to the circulation is not available to retard the decrease in plasma concentrations of the drug.

Propofol, (like thiopentone and alfentanil) has a short effect - site equilibration time.

There is no evidence of impaired elimination in patients with cirrhosis of the liver. Renal dysfunction also does not influence the clearance of propofol despite the observation that nearly three - fourths of propofol metabolites are eliminated in urine in the first 24 hours.

Propofol readily crosses the placenta, but is rapidly cleared from the neonatal circulation.

Volume of distribution	-	3.5 - 4.5 L/kg
Clearance	-	30 ml /kg/ min

Pharmacodynamics

Cardiovascular system

Propofol produces decreases in systemic blood pressure that are greater than those evoked by comparable doses of thiopentone. This decrease is often accompanied by decrease in cardiac output due to negative inotropic effect and decrease in systemic vascular resistance due to inhibition of sympathetic vasoconstrictor nerve activity. The hypotensive effects are generally proportional to the dose and the rate of administration of propofol, and may be potentiated by opioid analgesics.

Propofol blunts the pressor response to direct laryngoscopy and intubation more effectively than thiopentone. It also effectively blunts the hypertensive response to placement of a LMA.

Despite decreases in systemic blood pressure, heart rate often remains unchanged in contrast to the modest increases that typically accompany the rapid IV injection of thiopental. Bradycardia and asystole have been observed after induction. The incidence being 1.4 in 100,000.

Unlike sevoflurane, propofol does not prolong the QT_c interval on the ECG. It does not alter SA or AV nodal function in normal patients or in patients with Wolff - Parkinson- White syndrome.

Lungs

Propofol produces dose - dependent depression of ventilation, with apnoea occurring in 25% to 35% of patients after induction of anaesthesia with propofol. Opioids given as premedication enhance this

ventilatory depressant effect. A maintenance infusion of propofol decreases tidal volume and respiratory rate.

The ventilatory response to carbon dioxide and arterial hypoxemia are decreased by propofol. The decreased ventilatory response to hypercapnia is due to the effect at the central chemoreceptors. In contrast to low- dose volatile anaesthetics, the peripheral

chemoreflex response to carbon dioxide remains intact.

Propofol can produce bronchodilatation and decrease the incidence of intraoperative wheezing in patients with asthma. And propofol does not cause laryngospasm.

Central Nervous System

Propofol decreases cerebral blood flow, cerebral metabolic oxygen consumption and intracranial pressure. It also increases cerebrovascular resistance but does not appear to affect cerebrovascular reactivity to changes in arterial carbon dioxide tension.

Other systems

Propofol anaesthesia is associated with significant decreases in intraocular pressure by as much as 30 to 50 %. This decrease may be associated with a concomitant decrease in systemic vascular resistance.

Although propofol has the potential for affecting adrenal steroidogenesis, it does not appear to block cortisol and aldosterone secretion in response to surgical stress or adrenocorticotropic hormone (ACTH) in clinical practice. Although transient decreases in plasma cortisol concentrations have occurred, these reductions have not been sustained.

Side Effects

Allergic Reactions

Allergic components of propofol include the phenyl nucleus and di isopropyl side chain. Allergic reactions are described in patients with history of other drug allergies,

often to neuromuscular blocking drugs.

Lactic acidosis

Or "Propofol infusion syndrome" is described in patients receiving prolonged high-dose infusions of propofol (>75µg/kg/mnt) for longer than 24 hours. Unexplained tachycardia occurring during propofol anaesthesia should prompt evaluation.

Other side effects include

1. Abuse potential
2. Bacterial growth – the contents of an opened vial must be discarded if they are not used within six hours.
3. Pain on injection – 1ml of 1% lignocaine injection prior to propofol administration decreases the incidence.

Clinical uses

Induction of anaesthesia

The induction dose in healthy adults is 1.5 to 2.5 mg/kg IV, with blood levels of 2 to 6µg /ml producing unconsciousness depending on associated medications and the patient's age. Awakening typically occurs at plasma propofol concentrations of 1.0 to 1.5µg /ml.

k_{eo} is the rate constant for equilibration between plasma and the site of drug effect. (or) for transfer of drug from the site of drug effect to the environment.

$t_{½}$ k_{eo} for propofol is 2.4 minutes.

By knowing the k_{eo} of propofol, we can design a dosing regimen that yields the desired concentration at the site of drug effect.

Maintenance of anaesthesia

The typical dose of propofol for maintenance of anaesthesia is 100 to 300µg /kg/Minute IV, often in combination with a short acting opioid. General anaesthesia with propofol is generally associated with minimal post operative nausea and vomiting (PONV) and awakening is prompt with minimal residual sedative effects.

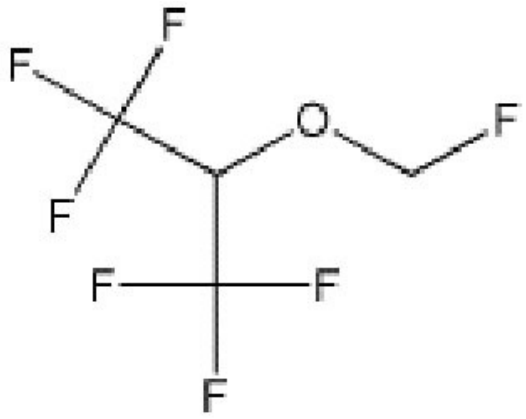
Antiemetic

Sub hypnotic doses of propofol are effective against chemotherapy induced nausea and vomiting when administered to induce and maintain anaesthesia, it is more effective than ondansetron in preventing PONV. Mechanism is unknown.

Antipruritic

Propofol, 10 mg IV, is effective in the treatment of pruritus associated with neuraxial opioids or cholestasis. The quality of analgesia is not affected by propofol.

PHARMACOLOGY OF SEVOFLURANE



Sevoflurane is fluorinated methyl isopropyl ether used for inhalational anaesthesia.

Physical Properties

Molecular Weight - 200 g/mol

Boiling point at 1 atm - 58.5˚C

Vapor pressure at 20˚C - 170 mmHg

Partition coefficient at 37˚C

Blood - Gas - 0.69

Brain - Blood - 1.7

Fat - Blood - 48

Oil - Gas - 47.2

MAC at P_B 760 mm Hg.

30-55 Years of age - 1.8%

For children

0-1 month	-	3.3%
1-6 months	-	3.0%
6 months - 3 years	-	2.8%
3-12 years	-	2.5%

MECHANISM OF ACTION OF INHALED ANAESTHETICS

Inhaled anaesthetics act in different ways at the level of the central nervous system. They may disrupt normal synaptic transmission by interfering with the release of neurotransmitters from presynaptic nerve terminal (enhance or depress inhibitory or excitatory transmission), by altering the re-uptake of neurotransmitters to the post synaptic receptor sites, or by influencing the ionic conductance change that follows activation of the post - synaptic receptor by neurotransmitters. Both, pre and post synaptic effects have been found.

Direct interaction with the neuronal plasma membrane is very likely, but indirect action via production of a second messenger also remains possible. The high correlation between lipid solubility and anaesthetic potency suggests that inhalation anaesthetics have a hydrophobic site of action. Inhalation agents may bind to both membrane lipids and proteins. It is at this time not clear which of the different theories are most likely to be the main mechanism of action of inhalation anaesthetics.

The Meyer - Overton theory describes the correlation between lipid solubility of inhaled anaesthetics and MAC and suggests that anaesthesia occurs when a significant number of inhalation anaesthetic molecules dissolve in the lipid cell membrane. The Meyer - Overton rule postulates that the number of molecules dissolved in the lipid cell membrane and not the type of inhalation agent causes anaesthesia. Combinations of different inhaled anaesthetics may have additive effects at the level of the cell membrane.

However, the Meyer - Overton theory does not describe why anaesthesia occurs. Mullins expanded the Meyer - Overton rule by adding the so-called Critical Volume Hypothesis. He states that the absorption of anaesthetic molecules could expand the volume of a hydrophobic region within the cell membrane and subsequently distort channels necessary for sodium ion flux and the development of action potentials necessary for synaptic transmission. The fact that anaesthesia occurs with significant increase in volume of hydrophobic solvents and is reversible by compressing the volume of the expanded hydrophobic region of the cell membrane supports Mullins critical volume Hypothesis.

The protein receptor hypothesis [postulates that protein receptors in the central nervous system are responsible for the mechanism of action of inhaled anaesthetics. This theory is supported by the steep dose response curve for inhaled anaesthetics. However, it remains unclear if inhaled agents disrupt ion flow through membrane

channels by an indirect action on the lipid membrane via a second messenger or by direct and specific binding to channel proteins.

Another theory describes the activation of Gama Amino Butyric Acid (GABA) receptors by the inhalation anaesthetics. Volatile agents may activate GABA channels and hyperpolarize cell membranes. In addition, they may inhibit certain calcium channels and therefore prevent release of neurotransmitters and inhibit glutamate channels. Volatile anaesthetics share therefore common cellular actions with other sedative, hypnotic or analgesic drugs.

The true mechanism of action of volatile anaesthetics may be a combination of two or more such theories described as multisite action hypothesis.

Pharmacokinetics

Uptake and distribution of inhaled anaesthetics:-

A series of partial pressure gradients, beginning at the vaporizer of the anaesthetic machine, continuing in the anaesthetic breathing circuit, the alveolar tree, blood and tissue will ensure the forward movement of the gas. The principal objectives of that movement are to achieve equal partial pressures on both sides of each single barrier. The alveolar partial pressure governs the partial pressure of the anaesthetic in all body tissues: they all will ultimately equal the alveolar partial pressure of the gas. After a short period of equilibration the alveolar partial pressure equals the brain partial

pressure. Alveolar partial pressure can be raised by increasing minute ventilation, flow rates at the level of the vaporizer and by using a non- rebreathing circuit.

Two special effects increasing the amount of gas in the alveoli have to be mentioned. The concentration effect describes how the concentration of the gas in the remaining alveolar volume can increase after some of the gas has been transferred into the blood. The second gas effect usually refers to nitrous oxide combined with an inhalation agent. Because nitrous oxide is not soluble in blood, its rapid absorption from alveoli causes an abrupt rise in the alveolar concentration of the other inhalation anaesthetic. All the above mentioned factors influence the inflow of gas into the alveoli.

Solubility, cardiac output, and the alveolar to venous anaesthetic gradient represent outflow factors. Inflow factors minus outflow factors equal alveolar partial pressure of the gas.

Solubility describes the affinity of the gas for a medium such as blood or fat tissue.

The blood:gas partition coefficient describes how the gas will partition itself between the two phases after equilibrium has been reached. The blood: gas coefficient of sevoflurane (0.69) ensures prompt induction of anaesthesia and recovery after discontinuation of the anaesthetic. Compared with isoflurane, recovery from sevoflurane anaesthesia is faster and the difference is magnified in longer duration

surgical procedures (>3 hours).

A higher cardiac output removes more volatile anaesthetic from the alveoli and lowers therefore the alveolar partial pressure of the gas. The agent might be faster distributed within the body but the partial pressure in the arterial blood is lower. It will take longer for the gas to reach equilibrium between the alveoli and the brain. Therefore, a high cardiac output prolongs induction time.

The alveolar to venous partial pressure difference reflects tissue uptake of the inhaled anaesthetics. A large difference is caused by increased uptake of the gas during the induction phase. This facilitates the diffusion of the gas from the alveoli into the blood. The brain: blood coefficient describes how the gas will partition itself between the two phases after equilibrium has been reached. Sevoflurane for example has a brain:blood coefficient of 1.7 meaning that if the gas is in equilibrium, the concentration in the brain will be 1.7 times higher than the concentration in the blood. All inhalation anaesthetics have high fat: blood partition coefficients. This enormous capacity of fat for anaesthetic means that most of the anaesthetic contained in the blood perfusing fat is transferred to the fat. Although most of the anaesthetic moves from the blood into the fat, the anaesthetic partial pressure in fat increases very slowly. The large capacity of fat and its low perfusion per milliliter explains the delayed recovery in obese patients.

PHARMACODYNAMICS

Central nervous system

Inhaled anaesthetics cause loss of response to verbal commands at MAC-awake

concentrations. Surgical stimulation increases the anaesthetic requirement to prevent awareness.

Volatile anaesthetics produce dose dependent increases in cerebral blood flow (CBF) .Sevoflurane has an intrinsic dose dependent cerebral vasodilatory effect. Sevoflurane does not alter auto regulation of CBF.

Inhaled anaesthetics produce increases in intracranial pressure that parallel increases in CBF produced by them.

Inhaled anaesthetics produce dose dependent decreases in cerebral metabolic oxygen requirements (CMR O_2). When EEG becomes isoelectric, an additional increase in the concentration of the volatile anaesthetics does not produce further decreases in CMR O_2.

Cardiovascular system

Volatile anaesthetics produce dose dependent decreases in mean arterial pressure. The decrease produced by sevoflurane principally results from a decrease in systemic vascular resistance.

Sevoflurane increases heart rate only at concentrations of >1.5 MAC. A small dose of opioid (morphine in the preoperative medication or fentanyl intravenously immediately before induction of anaesthesia) can prevent the heart rate increase associated with volatile anaesthetics.

Volatile anaesthetics exert little or no predictable effect on pulmonary vascular resistance.

Sevoflurane has no effect on the atrioventricular or accessory pathways and is considered an acceptable anaesthetic drug for patients undergoing ablative procedures.

Volatile anaesthetics induce coronary vasodilatation by preferential action on vessels with diameters from 20µ to 50 µ. They are cardio protective.

Respiratory system

Volatile anaesthetics produce dose dependent increases in the frequency of breathing and decreases in tidal volume. The net effect is a rapid and shallow pattern of breathing. The increase in frequency of breathing is insufficient to offset decreases in tidal volume, leading to decreases in minute ventilation and increases in $PaCO_2$.

Sevoflurane decreases the ventilatory response to carbon di oxide. It produces apnea between 1-5 and 2-0 MAC. All inhaled anaesthetics profoundly depress the ventilatory response to hypoxemia.

Sevoflurane produces bronchodilatation in normal individuals and in patients with COPD.

Skeletal Muscles

Sevoflurane and other fluorinated ethers produce skeletal muscle relaxation and produce dose- dependent enhancement of the effects of neuromuscular blocking drugs.

Obstetrics

Volatile anaesthetics produce dose dependent decreases in uterine smooth muscle contractility. This is desirable to facilitate removal of retained placenta. Conversely, uterine relaxation produced by volatile anaesthetics may contribute to blood loss due to uterine atony.

Metabolism

3 to 5 % of the dose administered undergoes oxidative metabolism by cytochrome P-450 enzymes to form organic and inorganic fluoride metabolites. In addition, sevoflurane is degraded by desiccated carbon dioxide absorbents containing strong bases to potentially toxic compounds especially when the temperature is increased. Among these compounds, only compound A- Triflurомethyl vinyl ether (and to a lesser extent compound B) are encountered clinically. Compound A is a dose dependent nephrotoxin in animals. Although this finding is a concern, the levels of these compounds (particularly compound A) that occur during administration of sevoflurane to patients are far below speculated toxic levels.

The reationale for utilizing at least 2L/minute of fresh gas flow rate when administering sevoflurane is intended to minimize the concentration of compound A that may accumulate in the anaesthesia breathing circuit to assess the adequacy of this recommendation, the concentration of compound A of anaesthesia with 1.25 MAC

sevoflurane during 2 to 8 hours was found to be in the range of 40 to 42 ppm which is far below the toxic levels.

In children, Sevoflurane anaesthesia lasting for 4 hours using total fresh gas flows of 2 L/ minute produced concentrations of compound A of <15 ppm, and there was no evidence of renal dysfunction.

A proposed mechanism of nephrotoxicity is metabolism of compound A via beta-lyase pathway to a reactive thiol. Because humans have less than one - tenth of the enzymatic activity for this pathway compared to rats, it is possible that humans should be less vulnerable to injury by this mechanism.

Probenacid is a selective inhibitor of organic anion transport and pretreatment with this drug prevents compound, A- induced renal injury in animals and may provide similar protection in humans.

Sevoflurane is non pungent, has minimal odour and causes the least degree of airway irritation among the currently available volatile anaesthetics. For these reasons, sevoflurane is acceptable for inhalation induction of anaesthesia.

ASSESSMENT OF RECOVERY AND HOME READINESS

For any day care procedure, the assessment of recovery is of paramount significance. A discrete time interval is no longer considered crucial for discharge; however, the patient must achieve clinical criteria that clearly reflect passage through the phases of early and intermediate recovery. The new short acting anaesthetics and analgesics have been instrumental in the faster recovery now seen after surgery. Some patients are now being transferred directly from the operating table to the step-down unit (phase II recovery) bypassing the PACU. This process is known as 'fast-tracking'.

STAGES OF RECOVERY

There are three stages of recovery following ambulatory surgery, namely Early, Intermediate and Late. Early and intermediate recoveries occur in the ambulatory surgical facility, whereas late recovery refers to the resumption of normal daily activities and occurs after discharge.

Early recovery is the time interval during which patients emerge from anaesthesia, recover their protective reflexes and resume motor activity. During this phase of recovery, patients are cared for in a phase I post anaesthesia care unit (PACU), where their vital signs and oxygen saturation are carefully monitored and supplemental oxygen, analgesia or antiemetics may be administered. The Aldrete score is commonly used to assess the fitness of patients to be transferred to the phase II recovery area.

Post Anaesthesia Recovery Score (Aldrete Score)

ACTIVITY

2 = Moves all 4 extremities voluntarily or on command

1 = Moves two extremities

0 = Unable to move extremities.

RESPIRATION

2 = Breathes deeply and coughs effectively

1 = Dyspnoeic

0 = apnoeic

CIRCULATION

2 = BP± 20 % of pre anaesthetic level

1 = BP± 21- 49% of pre anaesthetic level

0 = BP± 50 % of pre anaesthetic level.

CONSCIOUSNESS

2 = Fully awake

1 = Arousable on calling

0 = Not responding

OXYGEN SATURATION

2 = Maintains O_2 saturation> 92 % in room air.

1 = Needs O_2 to maintain saturation >90%

0 = O_2 saturation < 90 % with O_2 supplement.

Total score = 10

≥9 is needed for PACU Bypass.

The late recovery period starts when the patient is discharged home and continues until full, functional recovery is achieved and the patient is able to return to work. The surgical procedure itself has the highest impact on the full functional recovery.

Fast Tracking

The availability of rapid and short acting anaesthetic drugs for the maintenance of general anaesthesia has facilitated the early recovery of outpatients after ambulatory surgical procedures. Patients may be completely awake and oriented, breathing comfortably, with stable vital signs at the time they leave the operating room after brief ambulatory surgical procedures done under general anaesthesia. Significant cost savings may be achieved by bypassing the PACU. Care providers are the major cost of the PACU.

Assessment of Home Readiness

Guidelines for safe discharge from ambulatory surgical facility include stable vital signs, return to baseline orientation, ambulation without dizziness, no or minimal pain

and PONV, and minimal bleeding at the surgical site. All ambulatory surgical patients must have an escort to transport them home, and they must receive written postoperative instructions including advice on whom to contract in case a problem develops.

CAUSES OF DELAY IN DISCHARGE

Delays in discharge are typically related to persistent symptoms such as pain, PONV, dizziness, unsteady gait or, frequently, the lack of an escort. Excessive pain post operatively is a common surgery related cause of delayed discharge. Planning an appropriate prophylactic analgesic helps to eliminate the delay due to inadequate post operative pain relief.

PADSS (Post Anaesthesia Discharge Scoring system)

A discharge scoring system has been developed to evaluate and document patient's readiness for discharge objectively. The PADSS is a simple cumulative index that measures patient's home readiness and is based on five major criteria namely vital signs, activity, nausea & vomiting, pain and surgical site bleeding.

A maximum score of 10 is possible. Patients achieving a score of 9 or greater and have a responsible adult escort are considered fit for discharge. The requirement of patients to drink and void prior to discharge may not be necessary.

Modified PADSS

VITAL SIGNS (BP & PR)

2 = Within 20 % of preoperative baseline

1 = 20 % to 40% of preoperative baseline

0 = > 40 % of preoperative baseline.

AMBULATION

2 = Steady gait, no dizziness

1 = With assistance

0 = No ambulation or with dizziness

NAUSEA & VOMITING

2 = Minimal

1 = Moderate

0 = Severe

PAIN

2 = Minimal

1 = Moderate

0 = Severe

SURGICAL BLEEDING

2 = Minimal

1 = Moderate

0 = Severe

REVIEW OF LITERATURE

1. W.Scott Jellish et al. (1996)[6] did a study on the comparative effects of sevoflurane versus propofol in the induction and maintenance of anaesthesia in adult patients. They found that induction of anaesthesia time was significantly shorter with propofol than with sevoflurane. Emergence times after sevoflurane were significantly shorter than propofol. Overall frequency of complication-free induction, maintenance and emergence did not differ between the two anaesthetic groups. However, side effects involving airway excitement were more prevalent during mask induction with sevoflurane as compared to propofol. Sevoflurane compared favorably with propofol when used for anaesthesia for elective procedures of 1-3 hours duration.

2. Hanna Viitanen et al. (1999)[5] did a study on comparing the induction and recovery characteristics of sevoflurane anaesthesia induced with propofol or sevoflurane. They found that intubating conditions were similar in both groups. Emergence from anaesthesia occurred earlier with sevoflurane for induction than with propofol. The time to meet discharge criteria and recovery at home were similar.

3. Brain Fredman et al. (1995)[7] did a study on comparison of sevoflurane with propofol for outpatient anaesthesia. They found that induction of anaesthesia with propofol was significantly faster than inhalation induction with sevoflurane and there was no significant difference in the incidence of coughing, airway irritation or laryngospasm during induction of anaesthesia. Though the mean arterial pressures were similar, the use of sevoflurane was associated with consistently lower heart rate values during the early maintenance period. The time for fit for discharge was similar among the groups. But the use of sevoflurane for induction and / or maintenance of anaesthesia was associated with a higher incidence of postoperative emetic sequelae compared with propofol.

4. Hwan S. Joo et al. (1999)[10] did a meta analysis on sevoflurane versus propofol for anaesthetic induction. They found that sevoflurane and propofol had similar efficacy for anaesthetic induction. However, for routine outpatient surgery, propofol may still be the preferred induction anaesthetic because of its favorable induction characteristics, high patient satisfaction and less incidence of PONV.

5. J.K. Moore et al. (2003)[8] did a study on induction and recovery characteristics of propofol and halothane versus sevoflurane in paediatric day case surgery. They found that induction time was shorter with sevoflurane. Excitatory movement was more common during induction with sevoflurane. Incidence of delirium in recovery and PONV were higher in the sevoflurane group.

6. Reader. J et al.(1997)[13] did a study on recovery characteristics of sevoflurane or propofol based anaesthesia for day care surgery. They found that maintenance of anaesthesia with sevoflurane results in a more rapid emergence, but a higher incidence of nausea and vomiting compared with propofol. However the side effects were minor and did not result in any difference in time to discharge from the recovery ward or the hospital.

7. Chung, Frances, MD, FRCPC (1995) did a study and concluded that the post Anaesthesia Discharge scoring system (PADSS) is simple, practical, and easy to apply and to remember. In addition to permitting a uniform assessment of home readiness for patients, PADSS establishes a pattern of routine repetitive evaluation of patient's home readiness that is likely to contribute to improved patient outcome.

8. Leticia Delgado – Herrera et al. (2001)[4] in their review state that sevoflurane possess a number of characteristics that approach those of the ideal anaesthetic. Sevoflurane is versatile and can be used both as a mask induction inhalation anaesthetic and a maintenance agent for paediatric and adult surgical procedures.

9. V. Picard et al. (2000)[17] did a study on the quality of recovery in children: Sevoflurane versus propofol and they found that emergence and recovery times were comparable in both groups. There was a significant greater incidence of postoperative agitation in the sevoflurane group compared with the propofol group. This, did not, however delay discharge from the recovery room. The incidence of nausea and

vomiting was not significantly different among the groups.

10. Cynthia A. Lien et al. (1996)[14] did a study on comparison of the efficacy of Sevoflurane – Nitrous oxide or Propofol – Nitrous oxide for induction and maintenance of general anaesthesia in adults. They found that induction of anaesthesia was significantly slower in the sevoflurane group than in the propofol group. The ease of induction and the time required for emergence from the anaesthesia were same in both the groups. Patients in sevoflurane group experienced nausea and vomiting more frequently than patients in the propofol group which were not related to the administration of neostigmine or intraoperative opioids.

11. Julia C.F. Greenspun et al. (1995)[11] did a study on comparison of sevoflurane and halothane anaesthesia in children undergoing outpatient Ear, Nose and Throat surgery and found that sevoflurane anaesthesia had faster recovery than halothane anaesthesia in premedicated patients, but there was no difference in the time to meet home discharge criteria.

12. Sahar M. Siddik – Sayyid et al. (2005)[12] did a study on comparison of Sevoflurane – Propofol versus Sevoflurane or Propofol for Laryngeal Mask Airway insertion in adults and found that the co induction technique was associated with the most frequent incidence of successful LMA insertion at the first attempt than either

sevoflurane or propofol alone. Propofol induced anaesthesia allowed the fastest insertion of LMA and was associated with the least frequent incidence of PONV.

13. Anil Gupta et al. (2004)[18] did a systematic review on comparison of recovery profile after ambulatory anaesthesia with propofol, isoflurane, Sevoflurane and desflurane. They conclude that the differences in the early recovery times among the different anaesthetics were small and in favor of the inhaled anaesthetics. The incidence of side effects specifically post operative nausea and vomiting was less frequent with propofol.

14. Nicole Assman et al. (2004)[15] say that postoperative pain is one of the most common causes of delayed discharge and unplanned admission. PONV is a major cause of patient dissatisfaction with day care surgery. Opioid analgesics are important causative factors in PONV and therefore there is a trend away from using morphine (or at least to minimize the dose). Paracetamol or Non steroidal Anti inflammatory Drugs can be used.

15. Yaruz Guekan et al. (1999)[16] did a study on Propofol – Nitrous oxide versus Sevoflurane – Nitrous Oxide for strabismus surgery in children and found that the overall incidence of vomiting and antiemetic requirement in the first 24 hours was significantly higher in Sevoflurane – Nitrous oxide group than Propofol – Nitrous oxide group and the Propofol – Nitrous oxide group had significantly more episodes of oculo cardiac reflex than sevoflurane – nitrous oxide group.

16. K. R. Watson et al. (2000)[9] did a study on the clinical comparison of single agent anaesthesia with sevoflurane versus target controlled infusion of propofol. They found that propofol had a significantly faster induction time than sevoflurane but was associated with double the incidence of involuntary movements. Emergence times, characteristics, post operative nausea and vomiting and pain were unaffected by the anaesthetic technique. However, a more predictable emergence time was found following sevoflurane. Cardiovascular stability was good and comparable in both groups.

17. A. Thwaites et al. (1997)[19] did a study on inhalation induction with sevoflurane: a double - blind comparison with propofol. They found that induction of anaesthesia with sevoflurane was significantly slower when compared with propofol, but was associated with a lower incidence of apnoea and a shorter time to establish spontaneous ventilation. Induction complications were uncommon in each group but the transition to maintenance was smoother with sevoflurane and was associated with less hypotension compared with propofol. Emergence occurred significantly earlier compared with propofol.

18. Anton. A. van den Berg et al. (2005)[21] did an audit of preoperative patient preferences for intravenous or inhaled induction of anaesthesia in adults and found that 33% selected IV induction, 50% chose inhaled induction and 17% patients were undecided.

19. Mary E. Molloy, FFARCSI, Donal J. Buggy, MD, MSc, MRCPI, DME, FFARCSI, Patrick Scanlon, FFARCSI(1999) did a study on propofol or sevoflurane for laryngeal mask airway insertion and found that modified vital capacity breath inhalational induction with sevoflurane 8% is efficient for LMA insertion in most cases, but takes slightly longer than propofol.

20. Masaki Yurino et al. (1993)[20] did a study on comparison of spontaneous ventilation and vital capacity rapid inhalation induction (VCRII) techniques in induction of anaesthesia with sevoflurane, nitrous oxide and oxygen. They found that VCRII required only half the time of conventional inhalation induction and was not associated with cardiovascular instability. Each of the two techniques was found acceptable by most of the volunteers studied.

21. Suntheralingam Yogendran et al. (2005)[22] did a study comparing vital capacity and patient controlled sevoflurane inhalation induction and found that PCI was comparable to VCI in sevoflurane induction with respect to the speed of induction, side effects during induction and patient satisfaction. However, PCI requires no special training and is widely applicable to all patient populations

MATERIALS AND METHODS

This study was carried out in the ENT theatre, Government General Hospital,

Chennai after obtaining ethical committee and institutional approval. The aim of the study was to compare the induction and recovery characteristics of propofol and sevoflurane when they are used as single induction and maintenance anaesthetic agent in adult day care tonsillectomies.

STUDY DESIGN

The study was a randomized prospective study.

SELECTION OF CASES

Forty patients undergoing tonsillectomy were selected for the study. Their age ranged from 13 to 40 years. All the patients were assessed and those with normal clinical, biochemical, radiological and haematological parameters were selected. Informed written consent was obtained from all the patients and parents in case of minor. Each patient was randomly allocated to either the propofol or the sevoflurane group by lots. The groups were named 'P' for propofol and 'S' for sevoflurane.

INCLUSION CRITERIA

Assessed patients of ASA physical status I & II

Normal biochemical and haematological parameters

Age group between 13 to 40 years

No known hypersensitivity to egg or drugs

Airway – MPC I & II

Undergoing tonsillectomy and adenoidectomy
Surgery lasting around one hour

Patients normally able to ambulate well

Educated attender who can understand and carryout instructions.

EXCLUSION CRITERIA

Patient not willing

ASA class III and above

Patients with H/O drug or egg allergy

Anticipated difficult airway

H/O serious adverse experience with anaesthesia

Severe CVS/RS/CNS/ Metabolic disease

MATERIALS

1. Anaesthesia machine with sevoflurane vaporizer.
2. Appropriate drugs in labeled, preloaded syringes.
3. functioning Laryngoscope with appropriate size blades
4. Appropriate sized endotracheal tubes,
5. Equipments and drugs for resuscitation.

METHODS

Preoperative preparation

Patients were assessed pre-operatively. Procedure was explained to the patient and informed consent obtained. They were assessed with particular attention to any contraindications. The tests for recovery and the importance of strictly following instructions were emphasized.

Premedication

The patients were not given any IM premedication. No prophylactic antiemetic was given. All the patients received Glycopyrrolate 5µg/kg and Fentanyl 2 µg/kg just before induction of anaesthesia.

Conduct of Anaesthesia

On arrival of the patient in the operating room, monitors like pulse oximetry, NIBP and ECG were connected and baseline values of HR, BP and SPO2 were recorded. An intravenous access was obtained in the nondominant arm. 2% IV Lignocaine 1cc was given before induction to both the groups. Although lignocaine was given as prophylaxis against pain on injection of propofol, it was administered to both groups of patients because of possible effects on haemodynamic variables and to make it a constant.

PROPOFOL GROUP

The patients were induced with propofol 2mg/kg IV and intubated with 1.5mg/kg succinylcholine. After confirming and securing the endotracheal tube in position, they were connected to the closed circuit with nitrous oxide and oxygen in 2L: 1L ratio. Immediate post intubation, this group of patients received a continuous infusion of propofol 6-12mg/kg/hr (100-200 µg/kg/mnt) to maintain an adequate depth of anesthesia as judged by clinical signs and haemodynamic responses to surgical stimuli. Ventilation was controlled with vecuronium 0.8 mg/kg as the loading dose and one fourth of the loading dose as top up dose. They were given Diclofenac injection IM after intubation.

SEVOFLURANE GROUP

The patients are induced with sevoflurane 4% by patient controlled inhalation induction i.e. spontaneous ventilation (Penlon sigma Delta vaporizer) in Nitrous Oxide and oxygen in 4L: 2L ratio and intubated with 1.5mg/kg of succinylcholine. After confirming and securing the endotracheal tube in position, they were connected to the closed circuit with nitrous oxide and oxygen in 2L: 1L ratio with sevoflurane 1-2.5% to maintain adequate depth of anaesthesia. Ventilation was controlled with vecuronium 0.8mg/kg as loading dose and one fourth of the loading dose as top up dose. This group also received Diclofenac injection 1M after intubation.

Monitoring

Throughout the procedure, HR, ECG and SPO_2 were monitored continuously and NIBP was monitored every 5 minutes.

Recovery

Upon completion of the surgery, residual neuromuscular block was reversed with neostigmine 50µg/kg and glycopyrrolate 10µg/kg and then anaesthesia was discontinued. The patients' lungs were ventilated with 100% O_2 at a flow rate of 8L/mnt until tracheal extubation. The time of discontinuing the agent was taken as 'time zero' to calculate the recovery time.

Parameters studied

1. TIME TO LOSS OF CONSCIOUSNESS

Time interval from the start of induction to loss of eyelash reflex.

2. INDUCTION COMPLICATIONS

    1. Desaturation
    2. Coughing
    3. Laryngospasm
    4. Patient movement

3. INCIDENCE OF APNOEA

4. TIME TO PHASE I RECOVERY

This is the time taken from discontinuation of propofol or sevoflurane to the time when Aldrete score is ≥ 9.

5. TIME TO PHASE II RECOVERY

This is the time taken from discontinuation of propofol or sevoflurane to the time when the PADSS score is ≥ 9. It is also taken as the time to home readiness.

Statistical analysis

The descriptive statistics of the variables studied are represented as two-way

tables. The categorical factors are represented by the number and frequency (%) of cases. The continuous variables are represented by measures of central frequency (like mean, median) and deviation (say, standard deviation and range.) The differences in the properties are tested for statistical significance using non-parametric Chi-square test for variables measured on nominal scale. For variables measured on a continuous scale, when testing for two groups, Student "t" test is used to test for statistical significance in the differences of the two means.

OBSERVATIONS AND RESULTS

The patients included in the study were divided into two groups consisting of twenty patients each.

Group P (n=20) received Propofol Anaesthesia

Group S (n=20) received Sevoflurane Anesthesia

Table 1

Distribution of age of cases by groups[$]

Age	Group P	Group S	p-value
No. of cases	20	20	0.25
Mean	20.4	17.6	

S.D.	7.59	7.92	
Median	16.5	14	
Range	13 – 38	12 – 40	

$ Not statistically significant

The mean age was observed to be greater in Group P than Group S but not statistically significant.

Table 2

Distribution of cases by groups and sex$

Sex	**Group P (n=20)**		**Group S (n=20)**		**p-value**
	No.	**%**	**No.**	**%**	
Male	9	45.0	10	50.0	0.75
Female	11	55.0	10	50.0	

$ Not statistically significant

A female preponderance was forthcoming in Group P and equally distributed in Group S. The difference in the distribution between the two groups is not statistically significant.

Table 3

Distribution of weight of cases by groups[$]

Weight	Group P	Group S	p-value
No. of cases	20	20	
Mean	43.8	40.0	
S.D.	11.68	8.89	0.26
Median	40	40	
Range	30 – 60	30 – 60	

[$] Not statistically significant

The distribution of cases by weight and the difference in the mean values were observed to be not statistically significant between Group P and Group S.

Table 4

Distribution of cases by ASA and groups[$]

ASA	Group P (n=20)		Group S (n=20)		p-value
	No.	%	No.	%	
Grade I	20	100.0	20	100.0	1.00
Others	0	0.0	0	0.0	

[$] Not statistically significant

All the cases from both groups were identically classified as Grade I on ASA. Hence, there are no differences on ASA between the two groups.

Table 5

Distribution of cases by MPC and groups[$]

MPC	Group P (n=20)		Group S (n=20)		p-value
	No.	%	No.	%	
Grade I	16	80.0	19	95.0	0.34
Grade II	4	20.0	1	5.0	

[$] Not statistically significant

The distribution of number of cases by MPC and the two groups was not statistically significant (p=0.34) with more proportion of Grade I cases in among Group S than Group P.

Table 6

Distribution of cases by groups and MAP

MAP	Group P *(n=20)*		Group S *(n=20)*		p-value for difference of mean difference
	Actual	Difference from reference	Actual	Difference from reference	
PRE-OP[$]					
Mean	92.6	-	93.7	-	-
SD	9.42		8.38		
At induction					
Mean	80.6	-12.0	87.1	-6.6	0.12
SD	11.59	8.21	14.96	12.85	
POST-OP					
Mean	92.8	0.25	92.9	-0.75	0.80
SD	9.47	10.56	13.01	13.56	
At discharge					
Mean	88.0	-4.55	93.4	-0.30	0.07
SD	6.30	6.68	7.42	7.72	

[$] Reference category; Not statistically significant

The actual mean MAP values were generally lesser in Group P than Group S at all time points studied. The differences in the mean values of MAP at induction, Post-op and at discharge compared to the reference value at Pre-op between the two groups was observed to be statistically not significant.

Table 7

Distribution of cases by groups and pulse rate

Pulse rate	Group P *(n=20)*		Group S *(n=20)*		p-value for difference of mean difference
	Actual	Difference from reference	Actual	Difference from reference	
PRE-OP[$]					
Mean	91.6	-	97.3	-	
SD	11.88		15.39		
At induction					
Mean	105.8	14.2	98.8	1.6	0.02*
SD	11.52	10.85	25.19	21.34	
POST-OP					
Mean	89.4	-2.25	99.5	2.25	0.39
SD	12.71	10.94	17.48	20.49	
At discharge					
Mean	87.4	-4.2	95.3	-2.0	0.57
SD	9.54	9.45	11.15	14.44	

[$] Reference category; * statistically significant

The actual mean pulse rate values were generally lesser in Group P than Group S at all time points except at induction. The differences in the mean values at induction, Post-op and at discharge compared to the reference value at Pre-op between the two groups was observed to be statistically significant at induction and not at other time points.

Table 8

Distribution of time to LOC by groups

Time to location	Group P	Group S	p-value
No. of cases	20	20	
Mean	39.8	71.6	
S.D.	17.13	26.28	<0.001*
Median	35	75	
Range	20 – 90	20 – 140	

* Statistically significant

The mean time to LOC was observed to be lesser in Group P than Group S and the difference was statistically significant (p<0.001).

Table 9

Distribution of cases by incidence of apnoea and groups[$]

Apnoea	Group P (n=20)		GroupS (n=20)		p-value
	No.	%	No.	%	
No	2	0.0	2	10.0	1.00
Yes	18	90.0	18	90.0	

[$] Not statistically significant

There were equal number of cases with incidence of apnoea among both groups and the difference in distribution was statistically not significant.

Table 10

Distribution of cases by induction complication and group

Induction complication	Group P (n=20)		Group S (n=20)		p-value
	No.	%	No.	%	
Nil	19	95.0	13	65.0	0.04*
Yes	1	5.0	7	35.0	
Complication type					
Patient movement	0	0.0	3	15.0	
Bronchospasm	1	5.0	3	15.0	
Bradycardia	0	0.0	1	5.0	

* Statistically significant

The number of cases with induction complications was more among Group S than Group P and difference was statistically significant (p=0.04).

Table 11

Distribution of Phase I recovery by groups[$]

Phase I recovery profile	GroupP	Group S	p-value
No. of cases	20	20	
Mean	12	11	
S.D.	2.62	2.34	0.21
Median	11	10	
Range	8 – 17	8 – 17	

[$] Not statistically significant

The distribution of Phase I recovery profile between Group P and Group S is not statistically significant (p=0.21).

Table 12

Distribution of phase II recovery by groups

Phase II recovery profile	Group P	Group S	p-value
No. of cases	20	20	
Mean	105.5	97.5	
S.D.	11.11	12.06	0.10
Median	105	97.5	
Range	85 – 110	80 – 130	

[$]Not Statistically significant

The distribution of Phase II recovery profile between Group P and Group S is not statistically significant (p=0.01).

Table 13

Distribution of cases by post operative nausea/vomiting and group

Post operative nausea/vomiting	Group P (n=20)		Group S (n=20)		p-value
	No.	%	No.	%	
Nil	14	70.0	9	45.0	0.20
Yes	6	30.0	11	55.0	

$ Not Statistically significant

The distribution of post operative nausea / vomiting is less in Group P, but not statistically significant.

Table 14

Distribution of cases by post operative pain and group

Post operative pain	Group P (n=20)		Group S (n=20)		p-value
	No.	%	No.	%	
Nil	16	80.0	14	70.0	0.72
Yes	4	20.0	6	30.0	

$ Not Statistically significant

The distribution of post operative pain is less in Group P, but not statistically significant.

DISCUSSION

Intravenous agents are used commonly for induction of anaesthesia followed by inhalational agents for maintenance. A problem with this technique is the transition phase from induction to maintenance. The rapid redistribution of the intravenous agent could lead to lightening of anaesthesia before an adequate depth is attained with the inhalational agent. This has promoted the rediscovery of 'single agent' anaesthesia, which avoids problems associated with a transition phase.[9]

Propofol is a short acting general anaesthetic agent used widely for total intravenous anaesthesia because of its favorable recovery profile and low incidence of side effects. Propofol infusions are also becoming increasingly popular for maintenance of anaesthesia. However, use of propofol is associated with pain on injection, cardiovascular and respiratory depression and requires an intravenous drug delivery system [6, 7, 9]

Sevoflurane is a safe and versatile inhalational anaesthetic compared with currently available agents. Sevoflurane is useful in adults and children for both induction and maintenance of anaesthesia in inpatient and outpatient surgery. Of all currently used anaesthetics, the physical, pharmacodynamic, and pharmacokinetic properties of sevoflurane come closest to that of the ideal anaesthetic. These characteristics include inherent stability, low flammability, non – pungent odour, lack of irritation to airway, low blood: gas solubility allowing rapid induction of and emergence

from anaesthesia, minimal end-organ effects, minimal effect on cerebral blood flow, low reactivity with other drugs and a vapour pressure and boiling point that enables delivery using standard vapourisation techniques. [8] The availability of this agent makes it an alternative option for volatile Induction and Maintenance Anaesthesia (VIMA). [9]

Anton A. van den Berg, FRCA, Dudley A. Chitty, MD, Ramoun D. Jones, MD, Mir S. Sohel, MD and Ali Shahen, MD in their audit on preoperative patient preferences for induction of anaesthesia in adults found that 33% selected IV induction, 50% chose inhaled induction and 17% patients were undecided. They conclude that where manpower and facilities permit and in the absence of risk of regurgitation or airway difficulty, it is suggested that enquiry may be made of healthy adults presenting for elective ambulatory surgery as to their preferred route for the induction of anaesthesia. The inhalation induction done in our study was based on the above study.

A. Thwaites, S. Edmends and I. Smith in their study of inhalation induction with sevoflurane versus intravenous induction with propofol conclude that induction of anaesthesia with sevoflurane was significantly slower compared with propofol, but was associated with a lower incidence of apnoea and a shorter time to establish spontaneous ventilation.

Brain Fredman, MH. Nathanson, I. Smith, J. Wang, K. Klein and PF. White in their study of sevoflurane versus propofol was significantly faster than inhalation induction with sevoflurane and there were no significant difference in the incidence of coughing, airway irritation or laryngospasm during induction of anaesthesia.

In our study, we found that induction with sevoflurane is longer and associated with more complications. This is in concurrence with the study done by W. Scott Jellish, MD, PhD, Cynthia A. Lien, MD, H. Jerrel Fontenot, MD, PhD, and Richard Hall, MD, FRCPC, FCCPS comparing the induction and maintenance of anaesthesia in adult patients with sevoflurane and propofol. They found that induction of anaesthesia is

shorter with propofol. And side effects involving airway excitement were more during mask induction with sevoflurane as compared to propofol. This explains the more incidence of bronchospasm observed in the sevoflurane group.

The patient movement during intubation were slight movements of the hands or feet and did not compromise tracheal intubation or haemodynamics. [5] The observation of increase in the incidence of patient movement during induction with sevoflurane is supported by the study done by J.K. Moore, E.W.Moore, R.A. Elliott, A.S. St. Leger, K. Payne and J. Kerr on comparing the induction and recovery characteristics of propofol and sevoflurane.

Both propofol and sevoflurane produce dose dependent depression of ventilation and produce apnoea. Opioids given as premedication enhance this ventilatory depressant effort[2]. This explains the increased incidence of apnoea observed in both groups.

Though MAP decreased during induction of anaesthesia in both groups, the fall in MAP is more with induction of anaesthesia with propofol.[5] HR increased during induction of anaesthesia in both groups. This was probably due to the administration of glycopyrrolate prior to induction. [5,6]

The occurrence of bradycardia in one patient during induction of anaesthesia with sevoflurane could be explained by the direct sevoflurane induced inhibition of the beta-adrenoceptor system.[13]

Though statistically not significant, phase I recovery i.e. emergence from anaesthesia is shorter with sevoflurane than with propofol. This is in concurrence with the study done by A. Thwaites, S. Edmends and I. Smith on comparing the induction of anaesthesia with sevoflurane and propofol.

In our study, we found that the phase II recovery time after induction and maintenance of anaesthesia with propofol and sevoflurane were comparable. But the incidence of postoperative nausea and vomiting is more with sevoflurane anaesthesia and the number of patients complaining pain were more with sevoflurane anaesthesia. This observation is supported by the studies done by Brain Fredman et al (1995), Cynthia A Lien et al (1996), Reader. J et al (1997), Hanna Viitanen et al (1999) and V. Picard et al (2000).

The lower incidence of postoperative nausea and vomiting in the propofol group may be related to the 'intrinsic' antiemetic property of propofol. [7] The shorter time for requiring postoperative analgesics in the sevoflurane group probably reflects its rapid recovery profile and lack of tissue solubility and accumulation. It has been speculated that but not substantiated that propofol may have some analgesic effects. [6]

SUMMARY

Despite the low blood: gas solubility of sevoflurane, the inhalation induction of anaesthesia was slower than intravenous induction with propofol. Though the incidence of induction complications was more with sevoflurane group, they did not compromise tracheal intubation or haemadynamics except severe bradycardia observed in one patient.

The increased incidence of apnoea in both groups is attributable to the enhancement of the ventilatory depressant effect of propofol and sevoflurane by the opioid fentanyl .

The shorter emergence time in the sevoflurane group did not translate into a shorter hospital study. And the increased incidence of PONV and pain did not affect the

time for home readiness.

Though the small sample size in our study precludes drawing statistical conclusions, sevoflurane is found to be a useful alternative for elective procedures of short duration.

CONCLUSION

On comparing the induction and recovery characteristics of propofol and sevoflurane in adult tonsillectomies, it was found that:-

- Induction with sevoflurane is slower and with more complications.
- Incidence of apnoea is equal in both groups.
- Phase I & II recovery times were comparable between both groups.
- Sevoflurane anaesthesia was associated with high PONV and postoperative pain rate which is statiscally not significant.
- The smoother induction and less post operative PONV and pain with propofol make it more ideal for induction and maintenance of anaesthesia in adult outpatient surgeries.

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PROFORMA

DEPARTMENT OF ANAESTHESIOLOGY,

MADRAS MEDICAL COLLEGE, CHENNAI

COMPARISON OF INDUCTION AND RECOVERY CHARACTERISTICS OF PROPOFOL AND SEVOFLURANE IN DAY CARE ADULT TONSILLECTOMIES

1. Name :

2. Age/Sex :

3. IP NO/Unit :

4. PREOP

1. Comorbidity
2. Weight
3. Hb %
4. Blood Sugar
5. Blood Urea
7. PR
8. BP
9. SPO_2
10. CVS
11. RS

6. Serum Creatinine

12. ASA PS / MPC

Group S		Group P	
Drug	Dose given	Drug	Dose given
Glyco 5µg/kg		Glyco 5µg/kg	
Fent 2µg/kg		Fent 2µg/kg	
Sevoflurane 4% with 67% N_2O in 6L/mnt		Propofol .2mg/kg	
Succinyl choline 1.5 mg/kg		Succinyl choline 1.5 mg/kg	

VARIABLES ASSESSED:

1. Time to LOC in sec
2. Incidence of apnoea
3. Induction complications

Duration of procedure

Duration of Anaesthesia

Time since discontinuing sevoflurane / Propofol to recovery

Phase I (Aldrete /score ≥ 9)

Phase II (PADSS Score ≥ 9)

Time	MAP	PR	SPO_2
Preop			
Induction			
Maintenance			
1. 5 minutes			
2. 10 minutes			
3. 15 minutes			
4. 20 minutes			
5. 25 minutes			
6. 30 minutes			
7. 35 minutes			
8. 40 minutes			
9. 45 minutes			
10. 50 minutes			
11. 55 minutes			
12. 60 minutes			

MASTER CHART

SI.NO	AGE	SEX	WEIGHT	ASA	MPC	GROUP
1	26	F	50	I	I	P
2	31	M	60	I	I	P
3	25	F	40	I	I	P
4	27	M	60	I	I	P
5	15	F	35	I	I	P
6	22	F	45	I	I	P
7	15	M	30	I	I	P
8	14	M	30	I	I	P
9	15	M	30	I	I	P
10	13	F	30	I	I	P
11	15	F	30	I	I	P
12	18	F	40	I	I	P
13	15	F	50	I	II	P
14	13	M	40	I	I	P
15	30	M	60	I	II	P
16	14	F	35	I	I	P
17	38	F	60	I	I	P
18	18	M	50	I	I	P
19	30	F	60	I	II	P
20	14	M	40	I	II	P

SI.NO	AGE	SEX	WEIGHT	ASA	MPC	GROUP
21	14	F	40	I	I	S
22	26	F	50	I	I	S
23	15	M	50	I	I	S
24	13	M	30	I	I	S
25	16	M	35	I	I	S
26	13	M	30	I	I	S
27	13	M	30	I	I	S
28	13	F	40	I	I	S
29	14	M	30	I	I	S
30	13	F	30	I	I	S
31	35	F	50	I	I	S
32	13	M	35	I	I	S
33	16	F	40	I	II	S
34	18	F	40	I	I	S
35	40	F	60	I	I	S
36	13	M	40	I	I	S
37	13	M	50	I	I	S
38	26	F	50	I	I	S
39	13	F	35	I	I	S
40	15	M	35	I	I	S

INDUCTION PROFILE

SI.NO	TIME TO LOC IN SEC	INCIDENCE OF APNOEA	INDUCTION COMPLICATION	SI.NO	TIME TO LOC IN SEC	INCIDENCE OF APNOEA	INDUCTION COMPLICATION
1	90	Yes	NIL	21	75	Yes	Pt.movement
2	60	Yes	NIL	22	75	Yes	Pt.movement
3	40	Yes	NIL	23	75	Yes	Bronchospasm
4	40	Yes	NIL	24	52	Yes	NIL
5	75	Yes	NIL	25	90	Yes	NIL
6	30	Yes	Bronchospasm	26	60	Yes	Bronchospasm
7	70	Yes	NIL	27	60	No	NIL
8	45	Yes	NIL	28	60	Yes	NIL
9	40	Yes	NIL	29	75	Yes	NIL
10	20	Yes	NIL	30	50	Yes	NIL
11	30	Yes	NIL	31	75	Yes	Bronchospasm
12	30	Yes	NIL	32	120	Yes	NIL
13	35	Yes	NIL	33	90	Yes	NIL
14	30	Yes	NIL	34	60	Yes	NIL
15	25	Yes	NIL	35	20	Yes	Bradycardia
16	30	Yes	NIL	36	50	Yes	NIL
17	45	Yes	NIL	37	40	Yes	Bronchospasm
18	35	Yes	NIL	38	140	Yes	Pt.movement
19	30	Yes	NIL	39	90	No	NIL
20	35	No	NIL	40	75	No	NIL

MEAN ARTERIAL PRESSURE

SI.NO	PREOP	INDUCTION	POSTOP	DISCHARGE	SI.NO	PREOP	INDUCTION	POSTOP	DISCHARGE
1	74	61	94	82	21	98	97	85	100
2	86	79	91	81	22	97	124	76	83
3	99	90	109	96	23	82	82	77	89
4	84	79	89	86	24	99	95	111	88
5	88	78	90	84	25	99	67	90	109
6	95	72	75	97	26	102	78	99	95
7	103	83	98	94	27	107	95	101	99
8	97	80	92	85	28	91	71	70	90
9	87	79	102	92	29	105	104	99	100
10	86	83	88	86	30	75	57	84	81
11	80	61	78	79	31	81	80	116	93
12	92	93	98	88	32	99	94	93	99
13	110	95	87	91	33	96	98	99	99
14	101	87	87	86	34	86	97	89	97
15	100	77	106	90	35	90	78	83	86
16	80	56	81	73	36	90	75	105	99
17	105	101	111	97	37	99	85	115	100
18	95	93	92	89	38	94	90	79	86
19	90	88	94	91	39	97	96	99	90
20	99	74	94	93	40	86	79	88	84

PULSE CHART

SI.NO	PREOP	INDUCTION	POSTOP	DISCHARGE
1	86	108	92	82
2	94	90	77	80
3	105	116	116	98
4	89	100	93	80
5	92	98	86	84
6	103	120	92	89
7	77	96	78	74
8	97	118	76	80
9	87	104	96	94
10	113	123	112	108
11	91	113	97	94
12	74	124	98	82
13	96	105	90	90
14	91	102	78	90
15	77	88	70	78
16	98	102	92	104
17	113	116	107	86
18	92	102	78	83
19	68	84	76	74
20	89	107	83	98

SI.NO	PREOP	INDUCTION	POSTOP	DISCHARGE
21	97	113	76	102
22	81	86	84	84
23	99	102	84	88
24	117	129	88	102
25	102	93	78	96
26	110	111	111	83
27	87	92	110	84
28	102	76	77	86
29	73	118	90	96
30	95	82	114	93
31	70	88	123	103
32	124	118	108	108
33	118	142	121	114
34	108	124	117	98
35	86	24	92	78
36	84	76	110	106
37	104	102	105	96
38	82	88	75	78
39	115	110	130	116
40	91	102	97	94

RECOVERY PROFILE

SI.NO	DURATION OF ANAESTHESIA (IN MNTS)	PHASE i RECOVERY (IN MNTS)	PHASE II RECOVERY (IN MNTS)	SI.NO	DURATION OF ANAESTHESIA (IN MNTS)	PHASE i RECOVERY (IN MNTS)	PHASE II RECOVERY (IN MNTS)
1	48	10	110	21	60	9	90
2	26	16	120	22	55	10	95
3	28	14	125	23	40	11	105
4	40	11	110	24	52	10	100
5	50	11	100	25	55	12	110
6	76	12	110	26	32	9	85
7	65	15	130	27	28	10	95
8	47	9	100	28	50	10	100
9	60	11	110	29	45	12	95
10	75	17	105	30	55	14	125
11	40	8	95	31	48	17	100
12	66	14	110	32	74	13	130
13	57	10	100	33	44	8	80
14	47	9	95	34	35	9	100
15	50	15	110	35	24	10	95
16	60	12	100	36	41	12	105
17	74	11	85	37	30	8	85
18	55	15	90	38	76	11	95
19	38	10	105	39	42	10	100
20	37	10	100	40	36	15	95

PONV AND PAIN

SI.NO	PONV	PAIN
1	NO	NO
2	NO	NO
3	YES	NO
4	NO	YES
5	NO	NO
6	YES	YES
7	NO	NO
8	NO	NO
9	YES	NO
10	NO	NO
11	NO	NO
12	YES	NO
13	YES	YES
14	NO	NO
15	NO	NO
16	YES	NO
17	NO	YES
18	NO	NO
19	NO	NO
20	NO	NO

SI.NO	PONV	PAIN
21	YES	YES
22	NO	NO
23	YES	NO
24	YES	YES
25	NO	NO
26	YES	NO
27	YES	YES
28	YES	NO
29	NO	NO
30	NO	YES
31	NO	NO
32	NO	NO
33	YES	NO
34	NO	NO
35	YES	NO
36	NO	NO
37	YES	NO
38	YES	NO
39	YES	YES
40	NO	YES